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Health and Environmental Effects Profile for Alpha-Methylstyrene

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16. ABSTRACT

The Health and Environmental Effects Profile for alpha-Methylstyrene was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents. Quantitative estimates have been presented provided sufficient data are available. alpha-Methylstyrene has been determined to be a systemic toxicant. A Reference Dose (RfD), defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effect during a lifetime, for alpha-methylstyrene is 0.069 mg/kg/day for inhalation exposure. The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. The RQ value for alpha-methylstyrene is 1000.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ALPHA-METHYLSTYRENE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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LIST OF ABBREVIATIONS

BCF	Bioconcentration factor
BODT	Biochemical oxygen demand, theoretical
CNS	Central nervous system
CS	Composite score
GABA	γ-Aminobutyric acid
K_{oc}	Soil sorption coefficient
K_{ow}	Octanol/water partition coefficient
LD_{50}	Dose lethal to 50% of recipients
LOAEL	Lowest-observed-adverse-effect level
MED	Minimum effective dose
NOEL	No-observed-effect level
ppb	Parts per billion
ppm	Parts per million
RQ	Reportable quantity
RV_d	Dose-rating value
RV_e	Effect-rating value
SCE	Sister chromatid exchange
STEL	Short-term exposed level
TLV	Threshold limit value
TWA	Time-weighted average
UV	Ultraviolet
v/v	Volume per volume

DISCLAIMER

This document has been reviewed in accordance with U.S. Environmental Protection Agency policy and approved for publication. Mention of trade names or commercial products does not constitute endorsement or recommendation for use.

PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* (U.S. EPA, 1980) is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

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EXECUTIVE SUMMARY

alpha-Methylstyrene is a colorless liquid at ambient temperatures when pure (Hawley, 1981). It is soluble in a number of organic solvents but almost insoluble in water (Lewis et al., 1983; Weast, 1985).

The U.S. production of alpha-methylstyrene as an end-use product was 47.537 million pounds in 1983 (USITC, 1984). It is a by-product of cumene-phenol operations and is currently produced by six domestic manufacturers (Lewis et al., 1983; SRI, 1985). Virtually all of the alpha-methylstyrene commercially recovered is used as a monomer in the formulation of specialty polymers and resins (U.S. EPA, 1980a); ~40% of the U.S. production is exported (CMR, 1983).

If alpha-methylstyrene is released to the aquatic environment, volatilization is expected to be an important removal mechanism. The volatilization half-life from a river 1 m deep flowing at a speed of 1 m/sec, with a wind velocity of 3 m/sec, is estimated to be ~5 hours (Lyman et al., 1982). Although various pure culture biodegradation studies have shown that alpha-methylstyrene is oxidized by bacteria in natural water and in wastewaters (Dzhusupova, 1982; Ilyaletdinov et al., 1983; Dzhusupova et al., 1985; Grbic and Johanides, 1979; U.S. EPA, 1980a), the relative significance of microbial degradation in water cannot be determined from the available data. Hydrolysis, direct photolysis, oxidation by RO_2 radical and bioconcentration are not expected to be important aquatic fate processes. Oxidation by HO radical and singlet oxygen ($^1O_2$) in brightly sunlit waters may be important. Although alpha-methylstyrene has been detected in riverbed sediment (Steinheimer et al., 1981), it is not expected to partition strongly from the water column to sediments. If released to air in vapor-phase,

alpha-methylstyrene will react rapidly with photochemically produced HO radical (average estimated half-life of 4.5 hours) and with ozone (average estimated half-life of 24.7 hours). If released to soil, alpha-methylstyrene is expected to be subject to some leaching, based on estimated K_{oc} values of 134 and 650. Volatilization from soil surfaces may be an important process in its removal from soil.

alpha-Methylstyrene has been qualitatively identified in Philadelphia drinking water (Lucas 1984a,b), in Delaware River water (Sheldon and Hites, 1978), in riverbed sediment near Lake Charles, LA (Steinheimer et al., 1981), in groundwater located beneath pollution sources (Stepan et al., 1981; Botta et al., 1984) and in the urban atmosphere of six large Russian cities (Ioffe et al., 1979). Unidentified isomers of methylstyrene have been detected in Los Angeles air at 0.1-5.5 ppb (Grosjean, 1985), in the air of the Talladega National Forest, AL (Holzer et al., 1977) and in air contaminated by tobacco smoke (Holzer et al., 1976). Sources of alpha-methylstyrene exposure include wastewater effluents and air emissions from vents, tanks and process equipment involved in commercial alpha-methylstyrene production and use (U.S. EPA, 1980a). An estimated 25,019 U.S. workers are potentially exposed to alpha-methylstyrene yearly (NIOSH, 1984). Adequate data are not available to estimate the general population exposure to this chemical from inhalation of ambient air and ingestion of water and foods.

Little information is available concerning aquatic toxicity of alpha-methylstyrene. Concentrations of 2.00 and 0.20% (v/v) caused mortality and 0.02% inhibited population growth of rotifers, Dicranophorus forcipatus (Erben, 1978). Ogata et al. (1984) reported a BCF of 29.5 for goldfish. Yellow perch flavor was affected by exposure to 0.25 mg/ℓ for 168 hours (Persson, 1984).

alpha-Methylstyrene can be absorbed following oral, inhalation or dermal exposure (Bondareva, 1976; Aizvert, 1974, 1979), and it has been detected in the blood, liver and placenta of rats after oral dosing (Bondereva, 1976). The major metabolite in guinea pigs, rats and humans is atrolactic acid, which is excreted in the urine (Aizvert, 1974, 1975, 1979). Aizvert (1979) also reported that alpha-phenyl-alpha-hydroxypropanol glucuronide was a metabolite in humans. Excretion of urinary metabolites in humans followed first-order kinetics and was monophasic for the glucuronide and biphasic for atrolactic acid.

Pertinent data regarding the carcinogenicity of alpha-methylstyrene could not be located in the available literature as cited in the Appendix. A positive effect of alpha-methylstyrene was obtained in the SCE assay in human whole-blood lymphocytes (Norppa and Vainio, 1983).

Hardin et al. (1981) observed increased resorptions and changes in fetal sex ratio in the litters of rat dams injected intraperitoneally with 250 mg/kg methylstyrene (isomer not specified) during gestation. Teratogenic effects were absent. Increased embryonic mortality and teratogenic effects were reported in the offspring of rat dams exposed by an unspecified schedule to 5 mg/m^3 of alpha-methylstyrene by inhalation for 4 months (Serebrennikov and Ogleznev, 1978). Pertinent data regarding the reproductive effects of alpha-methylstyrene could not be located in the available literature as cited in the Appendix.

Subchronic inhalation exposure of rats and guinea pigs to alpha-methylstyrene at 600 ppm (2900 mg/m^3) or 800 ppm (3860 mg/m^3) for 7-8 hours/day, 5 days/week for up to 212 days or 28 days, respectively, resulted in slight changes in liver and kidney weights (rats at $\geq$600 ppm, guinea pigs at 800 ppm) (Wolf et al., 1956). No effects of treatment were observed in rats

and guinea pigs exposed to 200 ppm (965 mg/m³), 7-8 hours/day, 5 days/week for up to 149 days. Slight growth depression and slight mortality were observed in rabbits exposed to 600 ppm, 7-8 hours/day, 5 days/week for up to 212 days. Small sample size and inadequate reporting preclude comments concerning the significance of these results.

Biochemical changes in the brain were reported in rats exposed by inhalation to 40-70 mg/m³ alpha-methylstyrene either for 4-6 hours/day or continuously in foreign studies of 1-6 months duration (Solov'ev, 1974; Solov'ev and Barshkova, 1978; Klimina, 1974).

In many foreign studies of occupational exposure to alpha-methylstyrene or to mixtures of chemicals (alpha-methylstyrene, substituted benzenes, butadiene and other hydrocarbons) effects on the CNS system (Al'Berton et al., 1981; Sergeta et al., 1977), the respiratory system (Demchenko, 1978), liver (Konstantinovskaya, 1971; Sergeta et al., 1975) and the hematopoietic system (Putalova, 1979; Bravve, 1974; Maslov, 1982) were reported.

alpha-Methylstyrene is not highly toxic after acute oral exposure. The oral LD_{50} for rats was 4.9 g/kg (Wolf et al., 1956) and for mice was 5-10.25 g/kg (Ogleznev, 1964). Changes in neurotransmitter levels, amino acid content and enzyme activities in various organs in rats exposed to 3-5 mg/ℓ (3000-5000 mg/m³) for short periods (1-6 days) were reported by Klimina (1974) and Solov'ev and Barashkova (1978). Humans exposed to 600 ppm (2896 mg/m³) alpha-methylstyrene for a short time reported eye and nasal irritation as well as the detection of a strong odor (Wolf et al., 1956).

Rabbits are sensitive to the effects of dermally applied alpha-methylstyrene and exhibit erythema, inflammation, hyperemia, edema, exfoliation and sometimes necrotic changes of the skin (Wolf et al., 1956; Mirzoyan and

Zhakenova, 1972). When alpha-methylstyrene was placed in the eyes of rabbits, irritation of the conjunctival membranes was observed (Wolf et al., 1956).

Using an uncertainty factor of 1000, an RfD of 0.069 mg/kg/day or 4.83 mg/day for a 70-kg human was derived from a rat NOEL of 69 mg/kg/day, calculated from the exposure of 970 mg/m^3, 7.5 hours/day, 5 days/week in the subchronic inhalation study by Wolf et al. (1956). The higher exposure of 2900 mg/m^3, 7.5 hours/day, 5 days/week was the LOAEL and resulted in liver and kidney weight changes. The LOAEL was used to derive an RQ of 1000. Because data regarding carcinogenicity was not available, alpha-methylstyrene was classified as an IARC Group 3 and a U.S. EPA Group D chemical (U.S. EPA, 1986c).

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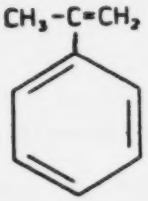
1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

alpha-Methylstyrene is the common chemical name, but this compound is also known as iso-propenylbenzene, (1-methylethenyl)benzene, b. a-phenylpropylene and 2-phenyl-1-propene. The structure, molecular weight, empirical formula and CAS Registry number for alpha-methylstyrene are as follows:



Molecular weight: 118.18

Empirical formula: C_9H_{10}

CAS Registry Number: 98-83-9

1.2. PHYSICAL AND CHEMICAL PROPERTIES

alpha-Methylstyrene is a colorless liquid at ambient temperatures when pure (Hawley, 1981). It is completely soluble in diethyl ether, benzene, acetone, carbon tetrachloride, ethanol, n-heptane and chloroform (Lewis et al., 1983; Weast, 1985). Selected physical properties are as follows:

Melting point:	-23.2°C	Lewis et al., 1983
Boiling point:	165.0°C	Lewis et al., 1983
Density at 20°C:	0.9106 g/cm³	Lewis et al., 1983
Refractive index at 20°C:	1.53864	Lewis et al., 1983
Vapor pressure:		
at 20°C	1.9 mm Hg	Lewis et al., 1983
at 25°C	2.5 mm Hg	Dreisbach, 1955
Water solubility at 25°C:	560 ppm	Lewis et al., 1983
Log K_{ow}:	3.27 (estimated)	U.S. EPA, 1986a
	3.35 (estimated)	Ogata et al., 1984

Flash point (open cup):	57.8°C	Lewis et al., 1983
Air odor threshold:	0.29 ppm	Amoore and Hautala, 1983
Water odor threshold:	0.043 ppm	Amoore and Hautala, 1983

alpha-Methylstyrene is subject to polymerization by heat or catalysts; the commercial product usually contains a polymerization inhibitor such as tert-butyl catechol (Hawley, 1981).

1.3. PRODUCTION DATA

The production of alpha-methylstyrene in the United States as an end-use product was 47.537 million pounds in 1983 (USITC, 1984). In addition, alpha-methylstyrene was produced as an unisolated intermediate for poly-methylstyrene by Amoco Chemical Corp. at a facility in Texas City, TX (SRI, 1985; Lewis et al., 1983). The current U.S. manufacturers of alpha-methyl-styrene are listed in Table 1-1.

Currently, all alpha-methylstyrene produced in the United States is as a by-product of cumene-phenol operations (SRI, 1985; Lewis et al., 1983). In these operations, cumene is oxidized to cumene hydroperoxide which is cleaved to phenol and acetone; however, as much as 2 wt% of the cumene may be converted to alpha-methylstyrene during the cleavage. The alpha-methyl-styrene is generally recycled, but it is also isolated as an end-product. Until 1977, Dow Chemical produced alpha-methylstyrene by alkylation of benzene with propylene followed by dehydrogenation (Lewis et al., 1983).

1.4. USE DATA

alpha-Methylstyrene has the following use pattern (CMR, 1983):

ABS resins	34%
Coatings (including adhesives)	14%
Polyester resins	4%
Miscellaneous	8%
Exports	40%

TABLE 141

U.S. Manufacturers of alpha-Methylstyrene
as of January, 1985*

Manufacturer	Location	Annual Capacity (millions of pounds)
Allied Corp. Allied Fibers & Plastics	Frankford, PA	24
Apex Oil Co. Clark Chem. Corp.	Blue Island, IL	4
Georgia Gulf Corp.	Bound Brook, NJ Plaquemine, LA	6 9
Standard Oil Co. (Indiana) Amoco Chem.	Texas City, TX	29
Texaco	El Dorado, KS	3
United States Steel Corp.	Haverhill, OH	38
	TOTAL	113

*Source: SRI, 1985

Virtually all of the alpha-methylstyrene that is commercially recovered is used in the formulation of specialty polymers and resins. The ABS resins are used primarily in automobile applications. Copolymers of alpha-methylstyrene and methylmethacrylate have been approved for use in food applications (U.S. EPA, 1980a).

1.5. SUMMARY

alpha-Methylstyrene is a colorless liquid at ambient temperatures when pure (Hawley, 1981). It is soluble in a number of organic solvents but almost insoluble in water (Lewis et al., 1983; Weast, 1985).

The production of alpha-methylstyrene in the United States as an end-use product was 47.537 million pounds in 1983 (USITC, 1984). It is produced as a by-product of cumene-phenol operations and is currently produced by six domestic manufacturers (Lewis et al., 1983; SRI, 1985). Virtually all of the alpha-methylstyrene commercially recovered is used as a monomer in the formulation of specialty polymers and resins (U.S. EPA, 1980a); ~40% of the U.S. production is exported (CMR, 1983).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Hydrolysis. The hydrolysis of alpha-methylstyrene in water is expected to be acid-catalyzed with the formation of a tertiary alcohol product; however, hydrolysis is negligible at pHs near neutral and not expected to be environmentally important (U.S. EPA, 1980a).

2.1.2. Oxidation. The reaction of alpha-methylstyrene with alkyl peroxy radicals (RO_2 radical) was studied by Howard and Ingold (1968). On the basis of an experimental rate constant of ~20 M^{-1} sec^{-1} at 30°C (Howard and Ingold, 1968) and a RO_2 radical concentration in natural water of ~10^{-9} M (Mill and Mabey, 1985), a half-life for this reaction is estimated to be ~400 days. Therefore, this reaction is not expected to be a significant fate determining process in water.

Olefins, in general, react with HO radical in water and have a reaction half-life of ~13-14 days in brightly sunlit natural water (Mill and Mabey, 1985). Therefore, this oxidation reaction may have some minor importance in the removal of alpha-methylstyrene from water. Both substituted and unsubstituted olefins react with singlet oxygen ($^1O_2$) in water. The estimated half-life of this reaction is 7-8 days (Mill and Mabey, 1985). Therefore, this reaction may have some significance in determining the fate of alpha-methylstyrene in water.

2.1.3. Photolysis. The UV absorption spectrum of alpha-methylstyrene in a cyclohexane solution exhibits minor absorption >300 nm (Sadtler, 1961); therefore, direct photolysis is possible. No rate data, however, were located that would indicate the environmental significance of direct photolysis.

2.1.4. Microbial Degradation. Kharitonova and Sklovskaya (1967) measured a 5-day 50.6% BODT for alpha-methylstyrene, which the authors regarded as a relatively slow bio-oxidation.

Various pure culture biodegradation studies have been conducted with alpha-methylstyrene. As many as 20 strains of bacteria isolated from synthetic rubber production wastewaters were able to use alpha-methylstyrene as a sole source of carbon and energy, with various Pseudomonas sp. being the most active (Dzhusupova, 1982). Pseudomonas acidovorans oxidized alpha-methylstyrene to acetophenone, whereas two P. aeruginosa cultures completely assimilated the compound (Dzhuspova et al., 1985). Various strains of Streptomyces have been able to use alpha-methylstyrene as the sole carbon source with <94% degradation in a 24-hour incubation (Grbic and Johanides, 1979). Bacteria strains isolated from river water have been able to grow on alpha-methylstyrene (U.S. EPA, 1980a). Certain methylotrophic bacteria are capable of oxidizing alpha-methylstyrene (Haber et al., 1983); however, no quantitative data regarding the biodegradability of this compound in natural water were available.

2.1.5. Volatilization. Based on vapor pressure and water solubility at 25°C (see Section 1.2.), the Henry's Law constant for alpha-methylstyrene is estimated to be 6.9×10^{-4} atm·m³/mol. This value of Henry's Law constant suggests that volatilization may be significant from ambient water, particularly from shallow rivers (Lyman et al., 1982). Using the method of Lyman et al. (1982), the volatilization half-life of this chemical from a river 1 m deep flowing at a speed of 1 m/sec with a wind velocity of 3 m/sec is estimated to be ~5 hours. Therefore, volatilization may be the most significant loss process for alpha-methylsytrene from water.

2.1.6. Adsorption. Based on estimated K_{oc} values of 134 and 650, alpha-methylstyrene is not expected to partition strongly from the water column to sediments. The detection of alpha-methylstyrene in riverbed sediment near Lake Charles, LA (Steinheimer et al., 1981) indicates that aquatic adsorption may occur, however.

2.1.7. Bioconcentration. Using recommended regression equations (Lyman et al., 1982), the BCF values for alpha-methylstyrene, based on a log K_{ow} value of 3.27 and water solubility of 560 ppm, are estimated to be 180 and 17.4, respectively. Ogata et al. (1984) measured a BCF value of 29.5 in goldfish reared in an aqueous solution containing 1 ppm alpha-methylstyrene. These data suggest that bioconcentration in aquatic organisms is not significant.

2.2. AIR

The vapor-phase rate constant for the reaction between alpha-methylstyrene and HO radical at 25°C has been experimentally determined to be 5.3×10^{-11} cm³/molecule-sec (Bignozzi et al., 1981). Assuming an average atmospheric HO radical concentration of 8×10^{5} molecules/cm³ (U.S. EPA, 1986b), a reaction half-life of 4.5 hours is estimated. The reaction products expected include acetophenone and formaldehyde (Bignozzi et al., 1981).

The rate constant for the vapor-phase reaction of alpha-methylstyrene with ozone has been theoretically calculated to be 1.3×10^{-17} cm³/molecule-sec at 25°C (U.S. EPA, 1986b). Assuming an average atmospheric ozone concentration of 6×10^{11} molecules/cm³ (U.S. EPA, 1986b), a reaction half-life of 24.7 hours is estimated.

The combined half-lives for HO radical and ozone indicate a vapor-phase removal half-life of 3.8 hours at an atmospheric temperature of 25°C.

Removal by these mechanisms is expected to decrease somewhat with decreasing temperatures and to increase as the concentrations of OH radical and ozone increase in polluted air.

2.3. SOIL

Pertinent data regarding the chemical degradation of alpha-methylstyrene in soil could not be located in the available literature and only limited data were available regarding microbial decomposition. The U.S. EPA (1980a) reported that various soil bacteria strains were unable to grow on alpha-methylstyrene. Sufficient data are not available to predict the relative significance or lack of significance of alpha-methylstyrene biodegradation in soil.

Experimental soil leaching studies were not located. Estimation of K_{oc} from water solubility, using the regression equation of Kenaga (1980), yields a K_{oc} value of 134. Estimation of K_{oc} from molecular topology and the quantitative structure-activity relationship analysis method of Sabljic (1984) yields a K_{oc} value of ~650. These K_{oc} values suggest that alpha-methylstyrene may be moderately mobile in soil and that some leaching may occur.

The relatively high vapor pressure of alpha-methylstyrene (2.5 mm Hg at 25°C) indicates that evaporation from dry soil surfaces is likely to occur. In addition, volatilization from water is predicted to be relatively rapid (see Section 2.1.5.). Therefore, volatilization from soil surfaces may be an important process in the removal of alpha-methylstyrene from soil.

2.4. SUMMARY

If alpha-methylstyrene is released to the aquatic environment, volatilization is expected to be an important removal mechanism. The volatilization half-life from a river 1 m deep flowing at a speed of 1 m/sec, with a wind velocity of 3 m/sec, is estimated to be ~5 hours (Lyman et al., 1982).

Although various pure culture biodegradation studies have shown that alpha-methylstyrene is oxidized by bacteria in natural water and in wastewaters (Dzhusupova, 1982; Dzhusupova et al., 1985; Grbic and Johanides, 1979; U.S. EPA, 1980a), the relative significance of microbial degradation in water cannot be determined from the available data. Hydrolysis, direct photolysis, oxidation by RO_2 radical and bioconcentration are not expected to be important aquatic fate processes. Oxidation by HO radical and singlet oxygen ($^1O_2$) in brightly sunlit waters may be important. Although alpha-methylstyrene has been detected in riverbed sediment (Steinheimer et al., 1981), it is not expected to partition strongly from the water column to sediments. If released to air in vapor-phase, alpha-methylstyrene will react rapidly with photochemically produced HO radical (average estimated half-life of 4.5 hours) and with ozone (average estimated half-life of 24.7 hours). If released to soil, alpha-methylstyrene is expected to be subject to some leaching, based on estimated K_{oc} values of 134 and 650. Volatilization from soil surfaces may be an important process in its removal from soil.

3. EXPOSURE

3.1. WATER

alpha-Methylstyrene was qualitatively identified in drinking water samples taken in Philadelphia during February, 1976 (Lucas, 1984a,b). An undetermined isomer of methylstyrene was qualitatively detected in drinking water in the United Kingdom (Fielding et al., 1981). Sheldon and Hites (1978) qualitatively detected alpha-methylstyrene in a shore-water sample taken from the Delaware River in October, 1976, but did not detect it in grab samples taken from the river in the summer of 1976 or the winter of 1977. Steinheimer et al. (1981) qualitatively identified alpha-methylstyrene in riverbed sediment from the Calcasieu River near the heavily industrialized area of Lake Charles, LA.

alpha-Methylstyrene has been found in groundwater beneath pollution sources, indicating that it can leach in soil. Stepan et al. (1981) detected it in Australia in an aquifer running beneath an abandoned quarry used for organic waste dumping. Botta et al. (1984) detected the compound in Italy in groundwater ~30 m beneath a paint factory where organics were leaking from storage tanks into the surrounding soil.

alpha-Methylstyrene can be released into the water environment from process wastewaters generated during its production and its use in polymer manufacture (U.S. EPA, 1980a). In addition, leaching from solid waste dumping is a potential source of exposure to groundwater.

3.2. FOOD

An unidentified isomer of methylstyrene was detected in 2/12 samples of human breast milk taken from women in four cities in the United States (Pellizzari et al., 1982).

alpha-Methylstyrene was 1/83 compounds identified in refrigerator air and it was found to adsorb to food stored in the refrigerator (Dmitriev et al., 1985). The source of alpha-methylstyrene in the refrigerator was apparently migration from polymers used in the construction of the unit.

3.3. INHALATION

alpha-Methylstyrene has been qualitatively identified in the urban atmosphere of six large cities in the Soviet Union (Ioffe et al., 1979). Grosjean (1985) reported the detection of methylstyrene (isomers not identified) at a concentration of 0.1-5.5 ppb in the morning air of Los Angeles during Autumn 1981. Unidentified isomers of methylstyrene have also been found in the air of the Talladega National Forest, AL (Holzer et al., 1977) and in air contaminated by tobacco smoke (Holzer et al., 1976).

NIOSH (1984) estimated that 25,019 workers in the United States were potentially exposed to alpha-methylstyrene yearly. Breathing zone air concentrations of nondetectable (<1 ppb) to 583 ppb were monitored inside a U.S. polystyrene production plant (Samimi and Falbo, 1982). Levels of 0-5 $\mu g/m^3$ were found in the vulcanization area of an Italian tire-retreading factory (Cocheo et al., 1983). Worker exposure to alpha-methylstyrene ranged from 0.16-0.67 ppm in U.S. plants manufacturing polyurethane foam insulation (Alcarese and Reisdorf, 1980; Reisdorf and Haggerty, 1980).

alpha-Methylstyrene exposure to the atmosphere can result from emissions from vents, storage tank losses and other process equipment involved in its production and use (U.S. EPA, 1980a). Diesel exhaust has been cited as an emission source of alpha-methylstyrene (Graedel, 1978). The thermal degradation of polystyrene may produce alpha-methylstyrene as a volatile product (Schroeder et al., 1984).

3.4. DERMAL

Pertinent data regarding dermal monitoring could not be located in the available literature as cited in the Appendix. Dermal exposure risk is probably greatest to workers involved in alpha-methylstyrene production and use.

3.5. SUMMARY

alpha-Methylstyrene has been qualitatively identified in Philadelphia drinking water (Lucas, 1984a,b), in Delaware River water (Sheldon and Hites, 1978), in riverbed sediment near Lake Charles, LA (Steinheimer et al., 1981), in groundwater located beneath pollution sources (Stepan et al., 1981; Botta et al., 1984) and in the urban atmosphere of six large Russian cities (Ioffe et al., 1979). Unidentified isomers of methylstyrene have been detected in Los Angeles air at 0.1-5.5 ppb (Grosjean, 1985), in the air of the Talladega National Forest, AL (Holzer et al., 1977) and in air contaminated by tobacco smoke (Holzer et al., 1976). Sources of alpha-methylstyrene exposure include wastewater effluents and air emissions from vents, tanks and process equipment involved in commercial alpha-methylstyrene production and use (U.S. EPA, 1980a). An estimated 25,019 U.S. workers are potentially exposed to alpha-methylstyrene yearly (NIOSH, 1984). Adequate data are not available to estimate the general population exposure to this chemical from inhalation of ambient air and ingestion of water and foods.

4. PHARMACOKINETICS

4.1. ABSORPTION

alpha-Methylstyrene can be absorbed following oral inhalation or dermal exposure. Although quantitative data regarding absorption by the gastrointestinal tract were not available, Bondareva (1976) detected alpha-methylstyrene in the blood, liver and placenta of rats treated orally with 2 g/kg. Additional details were not provided in the abstract.

Aizvert (1974) exposed guinea pigs, rats and humans to 0.02-4.0 mg/ℓ alpha-methylstyrene by inhalation. The percentages of inhaled alpha-methylstyrene excreted as the metabolite atrolactic acid were 21% in guinea pigs, 15.7% in rats and 26.2% in humans. These results indicate some absorption of the chemical by inhalation.

Aizvert (1979) placed 0.1 mℓ alpha-methylstyrene in contact with human skin for 8-10 minutes and calculated that 19.5 mg/cm² was absorbed. Additional details were not provided in the abstract.

4.2. DISTRIBUTION

Bondareva (1976) gave rats oral doses of 2 g/kg of alpha-methylstyrene and observed its distribution to the blood, liver and placenta.

4.3. METABOLISM

Aizvert (1974) measured 0.5-2.4 mg % of atrolactic acid in the urine of workers occupationally exposed to 2-27 mg/m³ alpha-methylstyrene. No atrolactic acid was observed in the urine of humans and animals exposed to 0.005 mg/ℓ. Aizvert (1975) found atrolactic acid in the urine of workers at concentrations of 2.1-2.4 mg % in fitters and 1.6-1.8 mg % in apparatus operations. When alpha-methylstyrene was subcutaneously injected into albino rats and guinea pigs, 9.6 and 15.6%, respectively, was transformed to atrolactic acid. When albino rats, guinea pigs and man were exposed to

alpha-methylstyrene by inhalation, 15.7, 21 and 26.2% of the inhaled concentration, respectively, were transformed into atrolactic acid. No other details were provided. Aizvert (1979) also reported that alpha-phenyl-alpha-hydroxypropanol glucuronide was a metabolite in humans.

4.4. Excretion

Guinea pigs, rats and man exposed by inhalation to 0.02-4.0 mg/ℓ alpha-methylstyrene excreted 21, 15.7 and 26.2%, respectively, of the inhaled concentration as atrolactic acid (Aizvert, 1974). In a human dermal exposure experiment, the excretion kinetics of alpha-methylstyrene metabolites was a first-order process and was monophasic for the alpha-phenyl-alpha-hydroxypropanol glucuronide and biphasic for atrolactic acid (Aizvert, 1979).

4.5. SUMMARY

alpha-Methylstyrene can be absorbed following oral, inhalation or dermal exposure (Bondareva, 1976; Aizvert, 1974, 1979), and it has been detected in the blood, liver and placenta of rats after oral dosing (Bondereva, 1976). The major metabolite in guinea pigs, rats and humans is atrolactic acid, which is excreted in the urine (Aizvert, 1974, 1975, 1979). Aizvert (1979) also reported that alpha-phenyl-alpha-hydroxypropanol glucuronide was a metabolite in humans. Excretion of urinary metabolites in humans followed first-order kinetics and was monophasic for the glucuronide and biphasic for atrolactic acid.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of alpha-methylstyrene could not be located in the available literature as cited in the Appendix, and this chemical is not scheduled for testing by the National Toxicology Program (NTP, 1986).

5.2. MUTAGENICITY

Norppa and Vainio (1983) tested concentrations of 0-10 mM alpha-methylstyrene (98-99% pure) in the SCE assay in human whole-blood lymphocyte cultures. A positive effect was noted, but the mean number of SCEs/cell was not doubled in the range of concentrations tested because of extreme toxicity at higher doses.

5.3. TERATOGENICITY

Hardin et al. (1981) gave groups of 10-15 young pregnant Sprague-Dawley rats weighing 250-300 g intraperitoneal injections of 250 mg/kg methylstyrene (isomer not specified) in corn oil on days 1-15 of gestation. Control rats were injected with corn oil only. Signs of maternal toxicity, including reduced body weight gain and changes in relative or absolute organ weight gain, were absent in the treated dams. No histopathological changes in maternal tissues were observed. Fetal toxicity was indicated by an increased incidence of resorptions ($p<0.05$) and by a changed fetal sex ratio ($p<0.05$) with a decrease in female fetuses. Teratogenic effects were absent in litters from treated dams.

Serebrennikov and Ogleznev (1978) exposed female rats to the maximum permissible concentration [not specified, but possibly 0.005 mg/ℓ (5 mg/m^3) (Aizvert, 1974)] of alpha-methylstyrene by inhalation for 4 months.

The exposure schedule was not further described. Embryonic mortality increased from 7.5% (presumably the control value) to 33.3% with exposure to alpha-methylstyrene. Teratogenesis increased from 3.0% (presumably the control value) to 21% in the offspring from treated dams. Additional details were not provided in the abstract.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of alpha-methylstyrene could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the effects of long-term oral exposure to alpha-methylstyrene could not be located in the available literature as cited in the Appendix. Wolf et al. (1956) exposed groups of 10-25 male and female Wistar rats and groups of 5-10 male and female guinea pigs to concentrations of 200 ppm (970 mg/m^3) and 600 ppm (2900 mg/m^3) alpha-methylstyrene for 7-8 hours/day, 5 days/week for 197 and 212 days, respectively, and to 800 ppm (3860 mg/m^3) and 3000 ppm (14,490 mg/m^3) for 7-8 hours/day, 5 days/week for 38 and $\leq$4 days, respectively. Groups of 1-2 rabbits and monkeys of either sex were exposed to 200 and 600 ppm for 7-8 hours/day, 5 days/week for 197 and 212 days, respectively. Surviving animals were sacrificed 18-22 hours after their last exposure. A limited histopathological examination of a few organs, and gross examination and weighing of the lungs, heart, liver, kidneys, spleen and testes were performed. No effects of treatment were observed in rats and guinea pigs exposed to 200 ppm; however, at 600 and 800 ppm, a slight change (presumably increase) in liver weight (absolute or relative, not specified) was observed. A slight change in kidney weight was observed in rats at concentrations of 600 and 800 ppm

and in guinea pigs at 800 ppm. Growth declined slightly in rats and guinea pigs at 800 ppm, and high mortality was observed at 3000 ppm. With the exception of slight growth depression and slightly increased mortality at 600 ppm, no effects were observed in the rabbits. No effects were observed in the rhesus monkeys.

Many foreign studies concerning toxicity of alpha-methylstyrene in animals were available in the published literature; U.S. EPA (1980a) reviewed the pre-1970 studies. Some representative post-1970 foreign studies include a report by Solov'ev (1974), who exposed rats to 0.05 mg/ℓ (50 mg/m³) of alpha-methylstyrene by inhalation for 4-5 hours/day (presumably daily) for 6 months. Accumulation of large amounts of NH_3, decreased total protein nitrogen and protein amide nitrogen were observed in the brain, as well as increases in glutamine formation and in the intensity of glycolysis. Other changes in brain tissue included decreased respiration and glycogen levels and the uncoupling of oxidative phosphorylation. Solov'ev and Barashkova (1978) observed changes in 13 cerebral amino acids after 4 months exposure to 0.05 mg/ℓ (50 mg/m³) alpha-methylstyrene for 6 hours/day in rats.

Klimina (1974) exposed rats to 0.04-0.07 mg/ℓ (40-70 mg/m³) methylstyrene by inhalation for 1-4 months. Two stages of toxicity were noted. The effects noted in the first stage were increased acetylcholine in the brain and kidneys, decreased acetylcholine in the blood and liver and inhibition of acetylcholinesterase in the blood and spleen. During the second or regenerative phase, acetylcholine and acetylcholinesterase levels were lower than normal in the liver and spleen, and blood and organ butyrylcholinesterase levels were elevated. The authors attributed these changes to adaptational and functional changes during alpha-methylstyrene exposure.

Makar'eva (1972) reported that rats and rabbits chronically exposed to alpha-methylstyrene and isopropylbenzene had decreased osmotic resistance of leukocytes and changed levels of neutrophils, glycogen, lipids and peroxidase.

U.S. EPA (1980a) summarized the pre-1970 foreign literature regarding the health effects from occupational exposure to alpha-methylstyrene. Representative post-1970 foreign studies are summarized in Table 5-1. In many of these studies, workers were exposed to other chemicals in addition to alpha-methylstyrene, including substituted benzenes, butadiene and other hydrocarbons. The major target organs, tissues and systems affected by exposure to these mixtures are the CNS, respiratory system, liver and hematopoietic system.

5.6. OTHER RELEVANT INFORMATION

Wolf et al. (1956) reported that 4.9 g/kg was the oral LD_{50} for alpha-methylstyrene in Wistar rats. Necropsy of the treated rats revealed slight liver changes and some kidney changes of questionable significance. U.S. EPA (1980a) reported oral LD_{50}s of 5 and 10.25 g/kg in mice (Ogleznev, 1964). Klimina (1974) exposed rats to a single inhalation of alpha-methylstyrene of 3-5 mg/ℓ (3000-5000 mg/m³) and observed decreased acetylcholine levels in the blood, kidneys and liver. Increased butyryl-cholinesterase was observed in the liver and kidneys, whereas in the brain and spleen butyrylcholinesterase was decreased. An increase in brain acetylcholinesterase activity and a decrease in acetylcholinesterase in the kidney was also observed. Solov'ev and Barashkova (1978) observed an increase of GABA, lysin, histidine and aspartate and a decrease in valine, methionine and tyrosine in the brains of rats exposed to 3-5 mg/ℓ (3000-5000 mg/m³) alpha-methylstyrene 6 hours/day for 6 days by inhalation.

TABLE 5-1

Human Health Effects of alpha-Methylstyrene*

Compound	Size of Exposed Population	Size of Control Population	Exposure Route	Level of Exposure	Duration of Exposure	Effect	Comment	Reference
alpha-Methylstyrene and divinyl	32 patients	25 unexposed subjects	occupational	NS	long-term	Neurocirculatory dystonia; increased kallikreinogen blood levels; reduced kallikreinogen, kininogen and kininase; disturbed liver function	NC	Al'berton et al., 1981
alpha-Methyl-styrene, iso-propylbenzene, benzene	102 workers	NS	occupational	NS	7-10 years	Increase in blood plasma bilirubin concentration; changes in enzyme activities and lipid metabolism	NC	Putalova, 1979
Divinyl-alpha-methylstyrene-rubber	NS	NS	occupational	NS	NS	Fatty and protein-producing functions of the liver were disturbed; disturbances in antitoxic, carbohydrate and pigment functions	NC	Konstantinovskaya, 1971
alpha-Methylstyrene	72 workers	controls were present, but size of group was not specified	occupational	NS	NS	Higher oxygen consumption and coefficient of utilization of oxygen	workers did hard manual labor while exposed	Demchenko, 1978
alpha-Methylstyrene	NS	controls were present, but group size was not specified	occupational	NS	1-5 years	Changes in the erythron system; vitamin B-12 level dropped to 352 µg/ml as compared with 578 µg/ml in controls; average erythrocyte diameter rose to 7.8 mµ; macrocyte percentage rose to 33; microcyte count was 10%; normocyte count was 57%	NC	Bravve, 1974

TABLE 5-1 (cont.)

Compound	Size of Exposed Population	Size of Control Population	Exposure Route	Level of Exposure	Duration of Exposure	Effect	Comment	Reference
alpha-Methylstyrene	NS	controls were present, but group size was not specified	occupational	NS	5-14 years	Changes in erythron system; vitamin B-12 level dropped to 203 µg/mℓ as compared with 578 µg/mℓ in controls; average erythrocyte diameter rose to 8.2 mµ; macrocyte percentage increased to 61; microcyte count was 10; normocyte count was 34%	NC	Bravve, 1974
alpha-Methylstyrene and hydrocarbons	exposed population of children living within 11 km of a petrochemical plant	control children lived 17 miles away where pollutant concentrations were below permissible limits for children	environmental pollution	above permissible limits	≤4 years	Higher number of erythrocytes (3.98×10^{12}-4.27×10^{12}/ℓ) in exposed children as compared with control children (3.65×10^{12}/ℓ); greater frequency and length of illness in exposed children; "adaptation mechanism of bloodstream strained"	NC	Maslov, 1982
alpha-Methylstyrene and butadiene	NS	NS	occupational	NS	NS	Increased ornithine carbamoyl transferase; some nervous disorders	NC	Sergeta et al., 1977
alpha-Methylstyrene divinyl	NS	NS	occupational	NS	"long time"	Changes in organ-specific enzyme activity observed before onset of chronic hepatitis	NC	Sergeta et al., 1975

*Sex of exposed and control populations was not stated.

NS = Not stated; NC = no comment

Additional information abstracted from the foreign literature concerning the acute toxicity of alpha-methylstyrene was reported in U.S. EPA (1980a). Wolf et al. (1956) reported that human subjects exposed to 600 ppm (2900 mg/m^3) alpha-methylstyrene for a short period had strong eye and nasal irritation and could detect a very strong odor of alpha-methylstyrene.

Wolf et al. (1956) applied alpha-methylstyrene to the ears and shaved abdomens of white rabbits 10-20 times over a 2- to 4-week period. Moderate to slight erythema and necrosis as indicated by a thin layer of devitalized tissue resulting in exfoliation was observed in the treated animals. alpha-Methylstyrene was not absorbed through the skin in acutely toxic amounts, as indicated by no observed changes in gross appearance, behavior and body weights. Mirzoyan and Zhakenova (1972) applied 30% alpha-methylstyrene to rabbit skin daily for 20 days and observed inflammation, hyperemia, edema and desquamation of the skin. Hyperkeratosis and changes in the thickness of the epidermal layer were also observed.

Wolf et al. (1956) placed two drops of alpha-methylstyrene into the right eyes of rabbits and examined them for signs of irritation and corneal damage from 3 minutes to 7 days after exposure. A slight, but perceptible irritation of the conjunctival membranes was observed without corneal injury in the treated rabbits.

5.7. SUMMARY

Pertinent data regarding the carcinogenicity of alpha-methylstyrene could not be located in the available literature as cited in the Appendix. A positive effect of alpha-methylstyrene was obtained in the SCE assay in human whole-blood lymphocytes (Norppa and Vainio, 1983).

Hardin et al. (1981) observed increased resorptions and changes in fetal sex ratio in the litters of rat dams injected intraperitoneally with 250 mg/kg methylstyrene (isomer not specified) during gestation. Teratogenic effects were absent. Increased embryonic mortality and teratogenic effects were reported in the offspring of rat dams exposed by an unspecified schedule to 5 mg/m³ of alpha-methylstyrene by inhalation for 4 months (Serebrennikov and Ogleznev, 1978). Pertinent data regarding the reproductive effects of alpha-methylstyrene could not be located in the available literature as cited in the Appendix.

Subchronic inhalation exposure of rats and guinea pigs to alpha-methylstyrene at 600 ppm (2900 mg/m³) or 800 ppm (3860 mg/m³) for 7-8 hours/day, 5 days/week for up to 212 days or 28 days, respectively, resulted in slight changes in liver and kidney weights (rats at $\geq$600 ppm, guinea pigs at 800 ppm) (Wolf et al., 1956). No effects of treatment were observed in rats and guinea pigs exposed to 200 ppm (965 mg/m³), 7-8 hours/day, 5 days/week for up to 149 days. Slight growth depression and slight mortality were observed in rabbits exposed to 600 ppm, 7-8 hours/day, 5 days/week for up to 212 days. Small sample size and inadequate reporting preclude comments concerning the significance of these results.

Biochemical changes in the brain were reported in rats exposed by inhalation to 40-70 mg/m³ alpha-methylstyrene either for 4-6 hours/day or continuously in foreign studies of 1-6 months duration (Solov'ev, 1974; Solov'ev and Barashkova, 1978; Klimina, 1974).

In many foreign studies of occcupational exposure to alpha-methylstyrene or to mixtures of chemicals (alpha-methylstyrene, substituted benzenes, butadiene and other hydrocarbons) effects on the CNS system (Al'Berton et al., 1981; Sergeta et al., 1977), the respiratory system (Demchenko, 1978),

liver (Konstantinovskaya, 971; Sergeta et al., 1975) and the hematopoietic system (Putalova, 1979; Bravve, 1974; Maslov, 1982) were reported.

alpha-Methylstyrene is not highly toxic after acute oral exposure. The oral LD_{50} for rats was 4.9 g/kg (Wolf et al., 1956) and for mice was 5-10.25 g/kg (Ogleznev, 1964). Changes in neurotransmitter levels, amino acid content and enzyme activities in various organs in rats exposed to 3-5 mg/ℓ (3000-5000 mg/m³) for short periods (1-6 days) were reported by Klimina (1974) and Solov'ev and Barashkova (1978). Humans exposed to 600 ppm (2896 mg/m³) alpha-methylstyrene for a short time reported eye and nasal irritation as well as the detection of a strong odor (Wolf et al., 1956).

Rabbits are sensitive to the effects of dermally applied alpha-methylstyrene and exhibit erythema, inflammation, hyperemia, edema, exfoliation and sometimes necrotic changes of the skin (Wolf et al., 1956; Mirzoyan and Zhakenova, 1972). When alpha-methylstyrene was placed in the eyes of rabbits, irritation of the conjunctival membranes was observed (Wolf et al., 1956).

6. AQUATIC TOXICITY

6.1. ACUTE

The only available information was provided by Erben (1978), who reported that a concentration of 2% (v/v) alpha-methylstyrene caused complete mortality of rotifers, Dicranophorus forcipatus, in 24 hours. Partial mortality occurred at 0.20% (v/v), and inhibition of population growth occurred at 0.02% (v/v) in 6-day exposures. Erben (1978), however, attributed these effects to oxygen depletion rather than to the direct effect of alpha-methylstyrene.

6.2. CHRONIC

Pertinent data regarding chronic toxicity of alpha-methylstyrene could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

Pertinent data regarding effects of alpha-methylstyrene on aquatic plants could not be located in the available literature as cited in the Appendix.

6.4. RESIDUES

Ogata et al. (1984) reported a BCF of 29.5 for goldfish, Carassius auratus.

6.5. OTHER RELEVANT INFORMATION

Persson (1984) reported that exposure to 0.25 mg/ℓ for 168 hours affected the flavor of yellow perch, Perca flavescens.

6.6. SUMMARY

Little information is available concerning aquatic toxicity of alpha-methylstyrene. Concentrations of 2.00 and 0.20% (v/v) caused mortality and 0.02% inhibited population growth of rotifers, Dicranophorus forcipatus (Erben, 1978). Ogata et al. (1978) reported a BCF of 29.5 for goldfish.

Yellow perch flavor was affected exposure to 0.25 mg/ℓ for 168 hours (Persson, 1984).

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

ACGIH (1985-1986) adopted a TWA-TLV of 50 ppm (~240 mg/m³) and a STEL of 100 ppm (~485 mg/m³) based on analogy to styrene (ACGIH, 1986). The OSHA (1984) ceiling limit is 100 ppm (480 mg/m³).

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of alpha-methylstyrene could not be located in the available literature as cited in the Appendix.

8. RISK ASSESSMENT

Pertinent data regarding the carcinogenicity of alpha-methylstyrene could not be located in the available literature as cited in the Appendix. This situation places alpha-methylstyrene in U.S. EPA Group D, meaning that there is inadequate evidence to assess its human carcinogenic potential. A positive effect of alpha-methylstyrene was obtained in the SCE assay in human whole-blood lymphocytes (Norppa and Vainio, 1983).

Hardin et al. (1981) observed increased resorptions and changes in fetal sex ratio in the litters of rat dams injected intraperitoneally with 250 mg/kg methylstyrene (isomer not specified) on days 1-15 of gestation, not accompanied by signs of maternal toxicity. Teratogenic effects were absent. Increased embryonic mortality and teratogenic effects were reported in the offspring of rat dams exposed by an unspecified schedule to 5 mg/m³ alpha-methylstyrene by inhalation for 4 months (Serebrennikov and Ogleznev, 1978). Pertinent data regarding the reproductive effects of alpha-methylstyrene could not be located in the available literature as cited in the Appendix.

In a subchronic toxicity study of alpha-methylstyrene, Wolf et al. (1956) exposed rats and guinea pigs to 600 ppm (2900 mg/m³) for 7-8 hours/day, 5 days/week for up to 212 days or to 800 ppm (3860 mg/m³), 7-8 hours/day, 5 days/week for 28 days and observed slight changes in liver weights. At concentrations of 600 and 800 ppm, slight changes in kidney weights were observed in rats, and in guinea pigs at 800 ppm. No effects of treatment were observed in rats and guinea pigs exposed to 200 ppm (965 mg/m³), 7-8 hours/day, 5 days/week for up to 149 days. Slight growth depression and slightly increased mortality were observed in rabbits exposed

to 600 ppm, 7-8 hours/day, 5 days/week for up to 212 days. Small sample size and inadequate reporting preclude comments concerning the significance of these results.

Biochemical changes in the brain were reported in rats exposed by inhalation to 40-70 mg/m³ alpha-methylstyrene either for 4-6 hours/day or continuously in foreign studies of 1-6 months duration (Solov'ev, 1974; Solv'ev and Barashkova, 1978; Klimina, 1974).

In many foreign studies of occupational exposure to alpha-methylstyrene or to mixtures of chemicals (alpha-methylstyrene, substituted benzenes, butadiene and other hydrocarbons), effects on the CNS (Al'Berton et al., 1981; Sergeta et al., 1977), the respiratory system (Demchenko, 1978), liver (Konstantinovskaya 1971; Sergeta et al., 1975) and the hematopoietic system (Putalova, 1979; Bravve, 1974; Maslov, 1982) were reported.

alpha-Methylstyrene has not been adequately tested for carcinogencity. Until adequate evidence is available regarding the carcinogenic effect of alpha-methylstyrene, it is appropriate to derive an RfD. Numerous abstracts concerning the effects of toxicity in animals and humans were available in the foreign literature but will not be considered in the risk assessment because the experiments are reported in summary form and do not provide the details necessary for quantitative risk assessment. The study by Wolf et al. (1956) was reported in enough detail to derive an RfD. The inhalation exposures in this study can be transformed into dosages by multiplying the concentration by 7.5 hours/24 days, 5 days/7 days, an absorption factor of 0.5 and the animal inhalation rate and by dividing by the animal body weight. Reference inhalation rates and body weights are 0.223 m³/day and 0.35 kg for rats (U.S. EPA, 1980b), 0.40 m³/day and 0.84 kg for guinea pigs, 2.0 m³/day and 3.8 kg for rabbits and 5.4 m³/day and 8.0 kg for

monkeys (U.S. EPA, 1985). At 2900 mg/m^3, 7-8 hours/day, 5 days/week for 212 days, rats (206 mg/kg/day) had increased liver and kidney weights, guinea pigs (154 mg/kg/day) had increased liver weights, rabbits (170 mg/kg/day) had slightly depressed growth rates and increased mortality, and monkeys (218 mg/kg/day) had no effects. No effects were observed in any species at 970 mg/m^3, 7-8 hours/day, 5 days/week for 197 days (69 mg/kg/day for rats, 52 mg/kg/day for guinea pigs, 57 mg/kg/day for rabbits and 73 mg/kg/day for monkeys). The doses corresponding to the 2900 mg/m^3 exposure level represent LOAELs for rats, guinea pigs and rabbits, and the doses corresponding to the exposure level of 970 mg/m^3 represent NOELs in all species. The transformed doses are essentially equivalent (the minor differences may be artificial because of assumptions made in the reference values); however, the best data were obtained for rats because the group sizes were larger (10-25 rats vs. 5-10 guinea pigs and 1-2 rabbits and monkeys). Dividing the rat NOEL of 69 mg/kg/day by an uncertainty factor of 1000 (10 for interspecies variability, 10 for intraspecies variability and 10 for subchronic data) results in an RfD of 0.069 mg/kg/day or 4.83 mg/day for a 70-kg human. This RfD is not highly recommended because Wolf et al. (1956) conducted an inhalation study, and uncertainties inherent in route-to-route extrapolation exist, because the effects reported (liver and kidney weight changes) are of questionable significance, and because it is preferable to use a study of longer duration (the lifetime of the animal) to calculate an RfD.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Wolf et al. (1956) was the only study available that reported sufficient data for calculating an RQ. This study was discussed in Section 5.5. and is summarized in Table 9-1. Although the effect in rabbits of slightly increased mortality appears to be the most severe effect, only "1 to 2 rabbits of either or both sexes" (Wolf et al., 1956) were used. Therefore, it is difficult to assess the significance of the "slight mortality," and a CS should not be calculated. Although the equivalent human dose was the same (see Table 9-1) for rats and guinea pigs, the rat data were chosen as the basis for the RQ because a smaller sample size of guinea pigs was tested and because more endpoints of toxicity were affected in rats (liver and kidney weight) than in guinea pigs (liver weight). The MED of 493 mg/day was calculated by multiplying the human equivalent dose by 70 kg and dividing by an uncertainty factor of 10 to approximate chronic exposure. The MED corresponds to an RV_d of 1.5 (Table 9-2). Changes in organ weight warrant an RV_e of 4. A CS of 6 was obtained by multiplying the RV_d by the RV_e giving an RQ of 1000 (Table 9-3).

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Pertinent data regarding the carcinogenicity of alpha-methylstyrene could not be located in the available literature as cited in the Appendix, placing alpha-methylstyrene in IARC Group 3 and in U.S. EPA Group D (U.S. EPA, 1986c).

TABLE 9-1

Inhalation Toxicity Summary for alpha-Methylstyrene[a,b]

Species/ Strain	Sex	No. at Start	Average Weight (kg)	Exposure	Transformed[c] Animal Dose (mg/kg/day)	Equivalent[d] Human Dose (mg/kg/day)	Response
Rats/ Wistar	M/F	10-25	0.35[e]	2900 mg/m³, 7.5 hours/day, 5 days/week for up to 212 days	412	70.5	Slight changes in liver and kidney weight
Guinea pigs/NR	M/F	5-10	0.84[f]	2900 mg/m³, 7.5 hours/day, 5 days/week for up to 212 days	308	70.5	Slight changes in liver weight
Rabbits/ NR	M/F	1-2	3.8[f]	2900 mg/m³, 5 days/week for up to 212 days	341	129	Slight decreased growth, death of unspecified number (mortality described as slight)

[a]Source: Wolf et al.,1956

[b]Purity of compound was not specified. Vehicle/physical state was vapor.

[c]Calculated by expanding over a 24-hour day and 7-day week and multiplying by animal inhalation rate of 0.223 m³/day for rats (U.S. EPA, 1980b), 0.40 m³/day for guinea pigs or 2.0 m³/day for rabbits (U.S. EPA, 1985) and dividing by the animal body weight.

[d]Calculated by multiplying the transformed dose by the cube root of the ratio of the reference body weight of the animal to that of a human (70 kg)

[e]Reference weight from U.S. EPA (1980b)

[f]Reference weight from U.S. EPA (1985)

NR = Not reported

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TABLE 9-2

Inhalation Composite Score for alpha-Methylstyrene in Wistar Rats[a]

Animal Dose (mg/kg/day)	Chronic[b] Human MED (mg/day)	RV_d	Effect	RV_e	CS	RQ
412	493	1.5	Slight change in liver and kidney weight	4	6	1000

[a]Source: Wolf et al., 1956

[b]An uncertainty factor of 10 was used to approximate chronic exposure

TABLE 9-3

alpha-Methylstyrene

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	inhalation
Dose*:	493
Effect:	slight changes in liver and kidney weight
Reference:	Wolf et al., 1956
RV_d:	1.5
RV_e:	4.0
Composite Score:	6
RQ:	1000

*Equivalent human dose

10. REFERENCES

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

ACGIH (American Conference of Governmental Industrial Hygienists). 1986. Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed. Cincinnati, OH.

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Windholz, M., Ed. 1983. The Merck Index, 10th ed. Merck and Co., Inc., Rahway, NJ.

Worthing, C.R. and S.B. Walker, Ed. 1983. The Pesticide Manual. British Crop Protection Council. 695 p.

In addition, approximately 30 compendia of aquatic toxicity data were reviewed, including the following:

Battelle's Columbus Laboratories. 1971. Water Quality Criteria Data Book. Volume 3. Effects of Chemicals on Aquatic Life. Selected Data from the Literature through 1968. Prepared for the U.S. EPA under Contract No. 68-01-0007. Washington, DC.

Johnson, W.W. and M.T. Finley. 1980. Handbook of Acute Toxicity of Chemicals to Fish and Aquatic Invertebrates. Summaries of Toxicity Tests Conducted at Columbia National Fisheries Research Laboratory. 1965-1978. U.S. Dept. Interior, Fish and Wildlife Serv. Res. Publ. 137, Washington, DC.

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